REMARKS

Claims 1-69 were originally presented in the present application. Claims 1, 3-12 and 70-72 are currently pending.

Amendments to the Claims

Applicant has currently amended Claim 1 and Claim 70. Applicant has further added new Claim 71 and new Claim 72.

Support for Claim Amendments

Support for each claim amendment can at least be found in the specification as originally filed as follows:

For new claims 71 and 72, support for "extending lifespan of a mouse cell or a human cell", can at least be found in the following sections of the specification as originally filed: Page 1, line 13-16; page 6, lines 11-13; and Page 16, line 10-11. Support for other elements of new claims 71 and 72 is as noted in the applicants Response of December 16, 2005.

Claim Rejection under 35 U.S.C. §112, first paragraph, Written Description Requirement (New Grounds of Rejection)

Examiner rejected previously pending Claims 1, 3-12, 15 and 70 under 35 U.S.C. § 112, first paragraph, alleging that the specification fails to comply with the written description requirement. Specifically, the Examiner states:

> Applicant has failed to (1) provide adequate written support to now claim "or a cell thereof" as it refers to mouse or human and (2) provide adequate written

support for the genus of pharmaceutically acceptable esters or amides of those C60 compounds presently claimed. (Page 3, paragraph 4).

With respect to the Examiner's allegation that the Applicants were not in possession of pharmaceutically acceptable esters or amides of the malonic acid compounds, Applicants first note that such embodiments of the invention are clearly supported by the specification. For example, see page 13, line 1-3 , which states: "...C_{60} compounds of the instant invention, which are referred to as carboxyfullerenes, have been mono- or multiply-derivatized with malonic acid, or the pharmaceutically acceptable malonic acid salts, esters and amides,...". Malonic acid is a carboxylic acid, and applicants note that pharmaceutically acceptable esters and amides of carboxylic acids were well known to those skilled in the art at the time of filing. In support of this position, applicants cite U.S. Patent 4,705,781, filed in 1986, as evidence that pharmaceutically acceptable esters and amides of carboxylic acids were well known at the time of filing. An inspection of the disclosure of columns 1 and 2 of U.S. Patent 4,705,781 clearly reveals that the identity and manner of making such esters and amides was well established in the pharmaceutical arts at least some six years before this application was filed. Given that pharmaceutically acceptable esters and amides of carboxylic acid compounds were well known at the time of filing, a person skilled in the art would in fact recognize that the Applicants had possession of the claimed invention. (See MPEP § 2163, citing Purdue Pharma L.P. v. Faulding Inc., 230 F.3d 1320, 1323, 56 USPQ2d 1481, 1483 (Fed. Cir. 2000) and Pfaff v. Wells Electronics, Inc., 55 U.S. at 66, 119 S.Ct. at 311, 48 USPQ2d at 1646). It is also well established that the written description requirement does not require that what is conventional and well known be disclosed in detail (See MPEP§ 2163, citing Hybritech Inc. v. Monoclonal Antibodies,

Inc., 802 F.2d at 1384, 231 USPQ at 94. and Capon v. Eshhar, 418 F.3d 1349, 1357, 76 USPQ2d 1078, 1085). Applicants therefore respectfully request that the examiner withdraw the written description rejections of pharmaceutically acceptable esters and amides of carboxylic acids in view of these considerations.

With respect to the Examiner's allegation that the Applicants were not in possession of methods of extending the lifespan of cells, Applicants first note that such embodiments of the invention are clearly supported by the specification. For example, see page 6, line 11, which states: "Another embodiment of the instant invention is a method of treating a metazoan comprising administering an antioxidant to the metazoan, which effectively increases the lifespan of that metazoan or the cells of that metazoan." Furthermore, applicants note that having demonstrated an increase in the lifespan of a metazoan in Example 2 of the specification, one skilled in the art would recognize that the Applicants were also in possession of an invention that increased the lifespan of cells of that metazoan. Such recognition by one skilled in the art would stem in part from the disclosure of U.S. Patent 6,265, 443 that was incorporated in its entirety in the specification (see page 5, lines 15 and 16). U.S. Patent 6,265, 443 revealed that the compounds of the invention inhibited neuronal cell death both in culture (Example 4 of the '443 patent) and in a well characterized transgenic mouse neuronal disease model (Example 6 of the '443 patent). One of skill in the art would also have recognized other studies demonstrating that the compounds of the invention decreased human cell death under certain cell culture conditions (see Monti et al. Biochemical and Biophysical Research Communications 277, 711–717 , 2000 reference in accompanying IDS). Recognition of possession would also have stemmed from the fact that organisms are made up of living cells and that the lifespan increases demonstrated by the

applicants in a whole organism were most likely accompanied by lifespan increases of the cells within that organism. Applicants therefore respectfully request that the examiner withdraw the written description rejections pertaining to extending the lifespan of cells in the new claims 71 and 72 in view of these considerations. In this regard, Applicants note that claim 1 has been amended such that it no longer encompasses "cells thereof" solely for the purpose of expediting prosecution and this amendment to claim 1 should not be construed as any admission that the Application does not provide adequate written description of this subject matter.

Claim Rejections under 35 U.S.C. §112, first paragraph, Scope of Enablement

Examiner rejected previously pending Claims 1, 3-12, 15 and 70 under 35 U.S.C. § 112, first paragraph, alleging that the specification, while being enabling for extending the lifespan of mice comprising the administration of C60 compounds, does not reasonably provide enablement for extending the lifespan of a human comprising the administration of the same.

Mice are an art-accepted models for extending lifespan.

In reference to the Roth et al. (Science, 305:1423, 2004) publication, the Examiner states: "...the state of the art is actually inviting further research and exploration into animal models to discover with greater genetic relevance to humans such that extrapolation of the results from the animal model to a human would logically follow." (Page 6, paragraph 3). Applicant agrees that better models are desirable to the study human aging. However, Applicant respectfully contends that the same holds true for all pre-clinical medical research. One can always *imagine* a better way of conducting research, however, the models systems that have been adopted as predictive in humans are those that necessarily take into account *practical* considerations.

Those skilled in the art appreciate that research is done in the most practical models available to the field. The question at hand is not what model theoretically is the most predictive, but rather what models are widely recognized in the art as having predictive value. Applicant respectfully reasserts the argument that what Roth et al. clearly and expressly states is that rodent models are, and have been, the most widely accepted for the study of aging with potential relevance to human aging and age-related disease. Below, Applicant will set forth a particularly relevant example from the art of the use of a mouse model to predict possible lifespan-extending benefits for humans.

Mice have been used to study potential life extending treatments in humans.

A specific example of the use of mice by skilled artisans in the study of potential life-extending treatments for humans was conducted by Baur and colleagues (Baur et al., Nature 444:337, 2006, provided in accompanying IDS). The Baur reference includes numerous investigators, skilled in the art of aging research, from diverse institutions including: the Gell Laboratories for the Biological Mechanisms of Aging, Harvard Medical School; the National Institute on Aging, National Institutes of Health; the Centre for Education and Research on Ageing, University of Sydney, Australia; and the Nutritional Neuroscience and Aging Laboratory, Pennington Biomedical Research Center, Louisiana State University System (Page 337, footnotes).

Resveratrol is a small molecule that has been "shown to extend the lifespan of evolutionarily distant species including *S. cerevisiae*, *C. elegans* and *D. melanogaster*" as well as in vertebrate fish (Baur et al., Page 337, paragraph 3). In their studies, Baur and colleagues examine the ability of resveratrol to improve the survival of mice on a high calorie diet. As

stated clearly in Baur et al., a high calorie diet is chosen for two reasons. First, obesity is a growing health concern in human populations, and therefore the study of aging in the context of obesity is very relevant to human health issues. Secondly, the use of a high calorie diet demonstrates that increases in aging can be achieved by administration of a small molecule, *independent of calorie restriction.*

Baur and colleagues selected resveratrol because it enhanced SIRT1 activity *in vitro*. SIRT1 has been implicated in mediating some of the physiological effects of caloric restriction in *S. cerevisiae* and *D. melanogaster* (Baur et al., Page 337, paragraph 2). "In mammalian cells, resveratrol produces SIRT1-dependent effects that are consistent with improved cellular function and organismal health" (Baur et al., Page 337, paragraph 3). Therefore, the authors hypothesized that resveratrol administration might recapitulate the lifespan extending benefits of caloric restriction without subjecting an organism to reduced calorie intake. Applicant respectfully notes the similarity between the Baur studies, that utilized a mouse model to demonstrate that a small molecule can extend lifespan without calorie restriction, and the Applicant's current invention that demonstrates that an unrelated small molecule can extend lifespan without calorie restriction.

Baur and colleagues chose to test concentrations in mice that were "feasible daily doses for humans" (Baur, *et al.*, Page 337, paragraph 4). The authors conclude their discussion by stating, "this study shows that an orally available small molecule at doses achievable in humans can safely reduce many of the negative consequences of excess caloric intake, with an overall improvement in health and survival" (Baur, *et al.*, Page 341, Discussion, paragraph 2). The Applicants pose the rhetorical question, "why choose concentrations achievable in humans?" and

respectfully assert that there would be no other reason to test concentrations feasible for humans, or to specifically point out such fact, if the mouse model had not been selected for its predictive value in humans. Clearly, the numerous authors contributing to the Baur et al. reference regarded mice as having predictive value with respect to humans.

The Kuro-o reference teaches that C57BL/6 mice are an art accepted model predictive of human aging.

In response to Applicant's previous assertion that "...the Examiner has not identified any defects in the experimental design that would question the truthfulness of Applicant's assertions" (Page 8, paragraph 1), the Examiner cites Kuro-o "Disease Model: Human Aging" (Trends in Molecular Medicine, April 2001). Examiner directs Applicant to the Table on page 180 (Table 1. Mouse models for human aging). Examiner states: "...Applicant's experiment upon which he relies as enabling support of lifespan extension in both mice and humans was performed in a C57B6 mouse, which is not one of the models recognized by Kuro-o as a potentially predictive animal model." (Page 8, paragraph 3). Examiner concludes by stating:

> It is clear that there is no evidence presented on the record or in the art that the particular mouse strain used in Applicant's experiment was suggested to have predictive efficacy in human aging. *The Examiner relies upon the teachings of Kuro-o in support of this conclusion.* Therefore, and in accordance with *In re Brana*, Applicant's mouse model is not accepted as correlating to human efficacy because the art specifically teaches away from such a conclusion. (Page 9, paragraph 1, emphasis added).

Examiner has confused the use of the term "model" in the context of a genetic mutation, and the use of the term "model" in the context of a model animal system. For instance, the

klotho mouse (kl-/-) is a mouse with a disruption in the klotho gene. On the other hand, C57B6 refers to a mouse *strain*, not a particular genetic mutation. One therefore can have a C57BL/6 mouse with a klotho mutation.

With the exception of the senescence-accelerated mice (SAM), which were developed by selective breeding of the AKR/J strain (Baur et al, Page 179, paragraph 3), the remaining examples reviewed by Kuro-o are of specific genetic mutations. These mutations have been created in laboratory mouse strains to generate mutant mice on a uniform genetic background. The C57BL/6 strain is the most commonly used laboratory inbred mouse strain (see http://jaxmice.jax.org/strain/000664.html; and http://aceanimals.com/C57BL6.htm; printouts in attached IDS). The C57BL/6 background is also the most often used in creating "knockout mice" (see http://www.genome.gov/15014549; printout in attached IDS). The klotho mouse was developed on a C57BL/6 background crossed with the C3H/J strain. The WRN mouse was also generated on a C57BL/6 background. Applicant's use of the C57BL/6 strain is therefore consistent with the models presented by the Kuro-o reference. Kuro-o does not teach away from using the C57BL/6 strain as a model for human aging, but instead gives several examples of its use.

The examples given by Kuro-o are of specific genetic mutations (genetic models) analyzed in a particular animal model (mouse strain). Applicant's experiments are not directed at a genetic model of aging, but rather at demonstrating that a particular compound can extend lifespan in an animal model predictive of human aging, i.e. an art-accepted mouse strain. Applicant respectfully refers Examiner to the Baur et al. reference. In their resveratrol experiments, Baur and colleagues also used C57BL/6 mice. In fact, Baur and colleagues

acquired their mice from the NIH's NIA Aging Rodent Colony, the same source of the Applicant's mice (Specification page 21, line 1-2). Therefore, Applicant's use of the C57BL/6 strain is consistent with others in the field doing research predictive of human aging, and entirely consistent with the Kuro-o reference that the Examiner "...relies upon...in support of this conclusion" (Page 9, paragraph 1).

As stated above, the Kuro-o reference is a review of specific genetic changes in mice that lead to various aging phenotypes. Applicant respectfully directs Examiner's attention to the fact that although all of the "models" set forth in the review are "mouse models", the title of the article is "Disease model:*human* aging." The title of the table on page 180 is "Mouse models for *human* aging." Clearly, Kuro-o is asserting that mouse models (including the C57BL/6 strain) are predictive of human aging. Thus, this reference bolsters Applicant's position that mice are an art accepted model predictive of human aging.

Examiner's Argument: *"The fact that the art recognized the unpredictability of animal models, particularly mice, of human aging is further evidence that the specification must contain more detail and guidance as to how to use the present invention in humans such that the skilled artisan would have been imbued with at least a reasonable expectation of success in achieving the lifespan extending effect as claimed without requiring an undue level of experimentation to determine how the results shown in the C57B6 mice were suggestive of the same level of efficacy in humans."*

The administration of a compound, such as C60, could be expected to extend longevity across species, including humans.

Examiner asserts that although calorie restriction works across species to extend longevity, C60 administration is a much more complex situation, and therefore one skilled in the art would not necessarily expect C60 to extend human lifespan. Examiner rejects Applicant's argument as "tenuous" that C60 administration could recapitulate the lifespan extending benefits of calorie restriction by acting through a similar mechanism, i.e. antioxidative activity (Page 9, paragraph 3 extending onto page 10). Examiner states that Applicant's remarks, "...fail to be persuasive because calorie restriction is a distinctly different process than the method substantially claimed in the present application." (Page 9, paragraph 3 extending onto page 10).

Applicant respectfully refers examiner to Baur et al. The premise of the experiments conducted by Baur and colleagues is that by targeting a pathway implicated in calorie restriction, one could achieve lifespan extension without a reduction in caloric intake. Thus, the premise behind the Baur reference is the exact same premise behind Applicant's current invention. A number of pathways have been implicated as mediating calorie reduction lifespan extension. In Baur et al., the authors chose to look at SIRT1 activation. In addition, Baur et al. states, "The ability of resveratrol to...modulate known longevity pathways suggests that resveratrol *and molecules with similar properties* might be valuable tools...." (Page 341, Discussion paragraph 1, emphasis added). In the current invention, the Applicants have focused on molecules that reduce oxidative stress, an art-recognized effect in the caloric restriction response:

> The current perception holds that the antioxidative action of [CR] seems far more widespread than earlier suspected...Many hypotheses, which were based on

epiphenomenal observations, could not be substantiated by the anti-aging action of [CR] and were, therefore, discounted. The oxidative stress theory is an outstanding exception that has endured [CR] scrutiny...Based on these findings, we have proposed that the ability to attenuate oxidative damage may be the major underlying mechanism of the anti-aging effect of [CR]" (from Yu, 1996, see also Masoro, 2005; Masoro, 2000; Sohal and Weindruch, 1996; Sohal et al., 1994).

The Baur et al. reference is a clear example that those skilled in the art believe that the lifespan extending effects of calorie restriction can be achieved independent of actual calorie restriction. This is despite Examiner's assertion that such means are "...significantly more complex than calorie restriction *per se.*" (Page 9, paragraph 3 extending onto page 10). This is because although the process of calorie restriction is distinct from administration of resveratrol or C60, it is not the act of calorie reduction, but the subsequent physiological response, that leads to lifespan extension. Therefore, by mimicking that response, a molecule such as resveratrol or C60 can be expected to lead to increased lifespan.

Examiner further contends that, "[c]alorie restriction can also be executed relatively simply and with minimal complexity, since the only requirement is that the total daily intake of calories is decreased." (Page 9, paragraph 3 extending onto page 10). However, Baur et al. states that "this study shows that an orally available small molecule at doses achievable in humans...with an overall improvement in health and survival" (Page 341, Discussion paragraph 2). Therefore, Baur et al. clearly indicates that one skilled in the art would predict that administration of a small molecule to achieve lifespan extension in humans is an achievable goal. In fact, the Applicants respectfully submit that administration of such a compound is a preferable

means in humans to severe caloric restriction. Methods of orally administering the C60 compounds in doses achievable in humans are also clearly disclosed by this application.

Examiner's Argument: *"[T]he presently claimed method is significantly more complex and requires the consideration of many factors, including, but not limited to, the determination of the dosage amounts needed, the frequency of administration, toxicology effects, pharmological and pharmacokinetic effects and the duration of administration."*

As noted above, the application provides ample guidance on dosage amounts as well as methods and timing of administration (see pages 14-16 of application as filed). Such determinations of dosing regimens are routine to those skilled in the art and thus need not be disclosed at an extraordinary level of detail as suggested by the examiner. Furthermore, the Examiners position that the Applicants must provide a detailed description of the toxicological, pharmacological and pharmacokinetic effects is simply not consistent with a large body of case law in this field that holds that utility and, by extension, enablement of pharmaceutical inventions should not hinge upon such demonstrations (see MPEP § 2107.03). Nonetheless, Applicants actually did provide evidence that C60 treatments were not toxic to rats (Example 3, page 22 of application as filed).

Examiner's Argument: *"Despite precedent that prior decisions did not require testing in humans, Examiner distinguishes the facts of this application to require more details."*

Applicant has put forth "sound scientific reasoning" as to why *in vivo* animal results are suggestive of human efficacy.

The Examiner states (Page 11, paragraph 2):

> ...[W]hile *it is acknowledged* that Applicants for patent are *not required to reduce the invention directly to practice in a human model* in order to claim the use of the therapy in humans...evidence *or soundly scientific reasoning* must be provided as to why one of ordinary skill in the art would have expected the in vivo animal results to be suggestive of the same or substantially similar efficacy in humans when the art clearly dictates to the contrary. (emphasis added).

Applicant refers Examiner to Baur et al. As stated previously, the Baur et al chose to use concentrations of resveratrol achievable in humans. The only rationale for this choice is that the mouse model was expected to be predictive of resveratrol administration to humans. The scientific reasoning is that resveratrol has been shown to increase lifespan across a host of species, and that it has been shown to target one of the pathways implicated in lifespan extension by calorie restriction. Thus, Applicant respectfully submits that this demonstrates "sound scientific reasoning...as to why one of ordinary skill in the art (Baur and colleagues)...expected the *in vivo* animal results to be suggestive of the same or substantially similar efficacy in humans."

Further, the Examiner has presented the Kuro-o reference that reviews seven different genetic mutations in mice, including mutations in the C57BL/6 background. Kuro-o explicitly presents these models as models of human aging. Thus, the art suggests that mice, including the C57BL/6 strain, are predictive of human aging.

C57BL/6 mice are not a calorie restricted strain of mice.

The Examiner states (Page 12, paragraph 2):

In response thereto, Applicant's attention is directed to the "Available Strains" from the National Institute of Aging (www.nia.nih.gov). In particular, it is noted that the C57BL/6 available from the National Institute of Aging is, in fact, contrary to Applicant's assertions, a calorie restricted mouse strain.

Applicant respectfully redirects Examiner's attention to the "Available Strains" from the National Institute on Aging (NIA). The NIA maintains its own colony of C57BL/6 mice to supply to researchers (as well as BALB/cBy, CBA/ and DBA/2 strains). *In addition*, the NIA maintains a colony of C57BL/6 that *have been calorie restricted.* These calorie restricted mice are available to researchers so that the researchers themselves do not have to raise and maintain a colony of calorie restricted mice, but can simply order them for experiments. The NIA does this for C57BL/6 mice because they are the most commonly used inbred strain. However, C57BL/6 mice are not a calorie restricted strain unless they have specifically been raised on a calorie restricted diet. Calorie restricted mice would be ordered by a researcher interested in studying the effects of calorie restriction. The Applicant was interested in studying the longevity of mice, but not longevity due to the effects of calorie restriction. Therefore, the Applicant's mice were of the C57BL/6 strain from the NIA, but not from the calorie restricted colony.

Whether a mouse strain is calorie restricted or not has to do with how the mice are raised, and has nothing to do with which strain of mice they are. Researchers ordering mice from NIA do not have to order calorie restricted C57BL/6 mice, and instead can order C57BL/6 mice that

have not been calorie restricted. Applicants respectfully refer Examiner to the Baur et al. reference. The mice utilized by Baur and colleagues were C57BL/6 mice from the NIA. In contrast to being calorie restricted, Baur and colleagues report that these mice were fed a standard diet or a high-calorie diet (Page 337, paragraph 4). This is consistent with Applicant's previous assertion that it is accepted practice in studies of aging to state whether animals or humans have been subjected to caloric restriction when reporting such data. The Applicant did not report any caloric restriction because none were imposed. Therefore, the Applicant has provided "factual evidence contrary to the assertion that the mouse strain used by Applicant is a calorie restricted strain."

Examiner's Argument: "*Use of inbred strain of mice does not preclude other genetic mutations.*"

Inbred strains of mice or other experimental organisms are used by researchers for the express purpose of avoiding the confounding effects of genetic variability in experiments. This is a commonly recognized fact and is also clearly evident from the description of the procedures used by the National Institute of Aging to maintain uniformity of the inbred mice colonies that they provide (see http://www.nia.nih.gov/ResearchInformation/ScientificResources/AgedRodentColoniesHandbook/ColonyMonitoringandHistory.htm; printout in attached IDS). While there is a very remote possibility that perhaps one or two of the inbred mice in the study somehow sustained a spontaneous mutation of some sort or other, the scenario proposed by the examiner where only the treated mice that exhibited longevity effects were comprised of mutants that increased

longevity while the untreated mice were wild type is essentially impossible. The Applicants subsequent reproduction of longevity effects in other experiments performed with inbred mice obtained from a distinct commercial source (Taconic Farms, Germantown, NY) renders the notion that the longevity effects were due to "genetic mutations" even more implausible (See Quick KL et al. Neurobiol. Aging Oct 30, 2006 e-pub in enclosed IDS). If the occurrence of confounding mutations in inbred mouse populations used in aging studies were a real concern, it would be unimaginable that the National Institute of Aging would provide such mice to researchers for aging studies or that peer reviewed scientific journals such as Neurobiology and Aging would publish experiments that used inbred mice. Should the Examiner choose to sustain this rejection based on this line of argumentation, the Applicants respectfully request that the Examiner produce some sort of evidence in the form of a peer reviewed scientific publication or other art-recognized authority that supports this position that genetic mutations in inbred mouse strains maintained by government or commercial sources for experimentation can confound experimental results.

Examiner's Argument: *"At this point, the invention would still require "undue experimentation" to work in humans."*

With respect to these arguments, the Applicants note that the specification provides a clear description of how to make and use the invention. More specifically, methods of administering the compounds, dose ranges of the compounds, dose forms of the compounds and dosing regimens are clearly disclosed by the specification (see pages 13 through 15). Given such information, those skilled in the pharmaceutical arts routinely set up clinical trials for testing the

effects of compounds in human patients. In this case, the endpoints for evaluating efficacy of the compounds in humans (i.e. lifespan increases) could, for example, be patterned after those reported for studies of calorie restricted humans (i.e. Biosphere 2 experiment in Walford et al., 1999; 1992 cited in the previous response). Many other examples of potential endpoints in aging studies can be found in the art. In considering whether experimentation is undue as alleged by the Examiner, the issue is not whether the experimentation is costly or time consuming but rather if the experimentation is routine (see MPEP § 2164.06 citing In re Wands, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404 and United States v. Telectronics Inc., 857 F.2d 778, 785, 8 USPQ2d 1217, 1223 (Fed. Cir. 1988), cert. denied, 490 U.S. 1046 (1989).). In this case, the Applicants have provided ample evidence that such experimentation is routine. The Examiner has in fact acknowledged as much in the previous Office Action in stating that "experimentation in this particular art is not at all uncommon". However, the Examiner has not explained why such experimentation would be undue in view of the admission that it is not uncommon. Consequently, the Applicant would respectfully request that the Examiner provide some evidence that such experimentation is non-routine and undue should the Examiner choose to sustain this rejection.

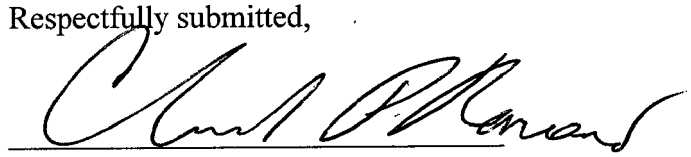
In view of the foregoing considerations, Applicants respectfully submit that one skilled in the art has ample scientific basis for believing that the instant invention, which disclosed the administration of an effective catalytic antioxidant that results in increased lifespan in mice, has enabled treatments for increasing the lifespan of humans and therefore respectfully request that this rejection be withdrawn.

CONCLUSION

All of the stated grounds of rejection have been properly traversed, accommodated, or rendered moot. Applicants respectfully request that the Examiner reconsider and withdraw each rejection. It is believed that a full and complete response has been made to the outstanding Office Action, and as such, the present application is in condition for allowance. If the Examiner believes, for any reason, that a personal communication will expedite prosecution of this application, she is invited to telephone the undersigned agent at the number provided.

Prompt and favorable consideration of this Response is respectfully requested.

Respectfully submitted,



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